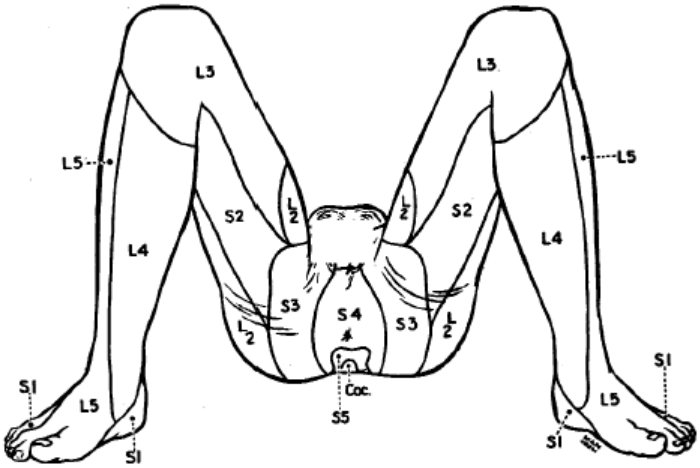
Neurogenic bladder
Preservation of renal function comes first

Anatomy revision

Parasympathetic motor (S234)	Lateral funiculus
	Lateral grey horn
	Pelvic nerves (nervi erigentes)
	Contraction of detrusor
	Inhibition of Onuf's nucleus
Sympathetic motor (T10-L2)	Lateral funiculus
	Lateral grey horn
	Hypogastric nerves
	Relaxation of detrusor
	Contraction of bladder neck
Somatic motor (S234)	Lateral funiculus
	Onuf's nucleus medial anterior horn
	Unconscious rhabdosphincter tone

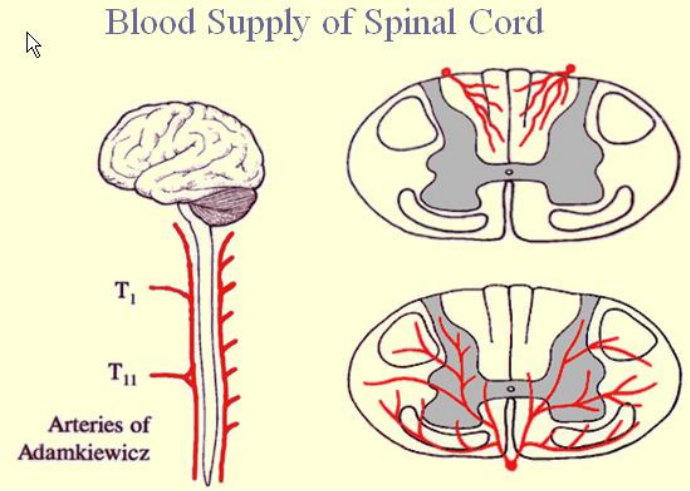
Spinal cord ends at at L1/2 vertebral junction. S2/3/4 sacral cord segments typically opposite T12. Therefore T11 lesions suprasacral, L1 lesions infrasacral, and T12 lesions variable



Reflexes (main levels)

Cremaster	L1/2
Knee	L3/4
Ankle	S1/2
Bulbocavernous	S3/4
Anal	S5

Terminology (ICS)
Overactive bladder – syndrome characterised by urgency with or without urge incontinence, usually with frequency and nocturia
Detrusor (bladder) overactivity – UDS diagnosis only; characterised by involuntary contractions during filling
Detrusor hyperreflexia – not a recommended phrase; replaced by neurogenic lower urinary tract dysfunction (NLUTD)



Classification systems

Multiple clasification systems proposed

Two most commonly utilised are;

(i) Location of defect

- Sacral or infrasacral
- Suprasacral
- Pontine or suprapontine

(ii) Functional outcome (i.e Madersbacher – recommended by EAU)

- Detrusor (overactive, normoactive or underactive)
- Sphincter (overactive, normoactive or underactive)

Expected behavior of the bladder and external sphincter in neurologic disease

Location of lesion	Clinical examination	Likely urodynamic findings
Suprapontine	Abnormal neurologic examination specific to condition	Detrusor hyperreflexia Synergic striated sphincter
Spinal/suprasacral	Muscle spasm, brisk reflexes	Detrusor hyperreflexia
	Positive digital anal reflex	Detrusor sphincter dyssynergia
	Positive bulbocavernosus reflex	Normal compliance
Conus or infrasacral	Negative digital anal reflex	Areflexic or underactive detrusor
	Negative bulbocavernosus reflex	Low compliance with open bladder neck Urethral sphincter incompetence

A. Suprapontine lesions

Failure of higher centre control leads to frequency, nocturia, urgency and urge incontinence. UDS show bladder overactivity with co-ordinated sphincter. Many older men may have co-existent bladder outflow obstruction – essential that UDS unequivocally diagnoses BOO before any surgery contemplated.

Common causes of suprapontine neurogenic bladder:

(i) Dementia

- Poorly understood neurological condition
- UDS in dementia

40% normal
40% bladder
15% SUI
5% retention with overflow

Bladder retraining impractical
Anticholinergics may exacerbate confusion (try trospium)
Surgical intervention for BOO poorly tolerated (pulled catheter etc.)
Timed voiding can help

(ii) Parkinson's disease

Basal ganglia disease – degeneration of substantia nigra
Relative dopamine deficiency vs. Ach
Dopamine believed to inhibit bladder contactions centrally
Urge and UUI exacerbated by poor mobility
UDS useful to exclude BOO, otherwise anticholinergics

(iii) Multiple-system atrophy

Aka Shy-Drager syndrome
Middle aged males
Autonomic neuropathy, esp basal ganglia, lateral horn cells and Onuf's nucleus
Postural HT, impotence and urinary incontinence
UDS – bladder overactivity and paralysed sphincter
No effective treatment: anticholinergics ineffective; catheterise when necessary

(iv) CVA

Acute phase a/w urinary retention; recovery characterised by bladder overactivity. UDS typically OAB with synergistic striated and smooth sphicter, occasionally normal. Typically urgency and urge incontinence. Basal ganglia and thalamus lesions tend to have preservation of levator function, therefore no incontinence

(v) Brain tumours

Variable
Posterior fossa tumours a/w retention of urine

(vi) Cerebral palsy

Typically normal tracts and normal voiding

B. Suprasacral lesions

Preserved spinal reflex arc; disruption of descending inhibition and pontine co-ordination. UDS typically show bladder overactivity with detrusor-sphincter dyssynergia. Usually normal compliance

(i) Suprasacral spinal cord injury

Initial injury characterised by spinal shock
Spinal shock
Withdrawal of inhibitory/excitatory influences 2' injury
Varible period (hours to weeks) characterised by hypocontractility, flaccidity and areflexia
Typically painless urinary retention with absent bulbocavernous and anal reflexes

Return of function often characterised by spasticity (bladder overactivity) and return of bulbocavernous and anal reflexes
Typically 6 months to 2 years

Detrusor sphincter dyssynergia
Failure of rhabdospincter to relax during bladder contraction
Initially no voiding, but bladder contraction strengthens to allow some leakage – voiding in staccato fashion
Seen in 70-100% of suprasacral spinal cord lesions
Risk factor for upper tract deterioration (see below)

Autonomic dysreflexia
Normally ascending nerve fibres carrying pain/distension signals send slips to sympathetic outflow and adrenal medulla in particular, preparing host for 'fight or flight'. Causes tachycardia, hypertension, headache, flushing, sweating and piloerection.
Peripheral baroreceptors trigger central parasympathetic response which act in 2 ways:
(i) Vagal outflow causing bradycardia
(ii) Descending inhibitory outflow to sympathetic nerve cell bodies, tempering sympathetic response
In lesions above T6 spinal inhibition does not occur, leading to uncontrolled hypertension. Vagal outflow is preserved explaining bradycardia
May be anticipated and painful stimuli avoided if possible. Spinal anaesthesia better vs. GA.
Treatment of established autonomic dysreflexia
Remove stimulus (i.e. draining bladder)
Sit up (lessen chance of hypertensive brain injury)
GTN spray/buccal suscard
Sublingual nifedipine or captopril
IV labetolol

Risk factors for upper tract deterioration
DSD
Low (poor) compliance < 20ml/cm water
VUR on video UDS
High DLPP > 40 cm water

Hydronephrosis seen in ~ 20% of spinal injuries patients at 15-20 yrs (Hackler 1977)

(i) <u>Spinal bifida (myelomeningocoele)</u>
See paediatric neuropathy
Classification (Rickwood and Thomas; Mundy)
Suprasacral (discoordinated voiding; aka contractile bladder)
Retained sacral conus reflexes (anocutaneous (S5); bulbocavernosus (S3,4))
Detrusor hyperreflexia
DSD
No sphincter paralysis

Sacral (acontractile bladder)
Absent sacral conus reflexes
Often poorly compliant bladder
High pressure (retainers) or low pressure (wetters) dependent on intrinsic EUS tone (static sphincteric obstruction)
NB. Paralysis of EUS means that intrinsic tone may be overcome by external pressure – 'expressible bladder' pathognomonic for neuropathic bladder
There may be an intermediate type characterised by detrusor overactivity and a weak external sphincter similar to Shy-Drager

(ii) Multiple sclerosis
Demyelinating disorder characterised by white plaques in CNS/cord
Commonest cord segment involved = cervical
Young adults; females > males; Northern latitudes
Relapsing remitting in 85%; 80-100% have LUTS
Typically detrusor overactivity and DSD. Irritative LUTS and impaired bladder emptying
DSD appears safe in MS* – very low risk of upper tract deterioration.
OK to teach ISC and discharge
* Holds true for all forms of progressive neurological disease

C. Sacral and infrasacral lesions
Usually characterised by bladder acontractility and paralysis of urethral sphincter. Residual tone at BN and EUS (? sympathetically mediated) overcome at varying degrees of bladder filling. Bladder emptied by abdominal strain or fist (Crede manouvre). Dependent upon DLPP (no contraction) upper tract may be at risk. If DLPP > 40cm water risk of upper tract deterioration. Examination reveals absent bulbocavernous and anal reflexes usually with flaccid areflexic legs.

(i) Sacral cord (conus) injury
Acontractile bladder with paralysed sphincter
Often poor bladder capacity +/- poor compliance – no cycling
Mx = improve capacity and re-inforce/close sphincter
(ii) Lumbar disc herniation
Typically L4/L5 or L5/S1 interspaces
Central disc protrusion a/w cauda equina syndrome
Cauda equina syndrome
Acute back pain, saddle anaesthesia, painless urinary retention and defeacation problems
Distended bladder, sensory loss, absent bulbocavernous and anal reflexes, lower limb flaccidity and areflexia
Give IV dexamethasone 16mg, arrange urgent MRI and refer to spinal surgeons
UDS findings most commonly normal compliance, areflexia and pelvic floor denervation
(iii) Pelvic surgery
Typically AP-resection, anterior resection and hysterectomy

NLUTD reported in up to 60% post-op; of these 15% permanent
Due to damage to hypogastric and pelvic nerves
UDS shows reduced sensation; hypocontractility, poor complinace and paralysis of pelvic floor
Spontaneous recovery in 85%; reassure and teach ISC

(iv) Diabetes mellitis

Chronic hyperglycaemia results in autonomic and PN demyelination
Diabetic NLUTD may be predicted by the presence of peripheral neuropathy: 75-100% of patients with sacral dermatome parasthaesia have bladder dysfunction. Sacral reflexes may be reduced
UDS shows impaired sensation, hypocontactility and high PVR

(v) Other

Herpes zoster	VZV; sacral dermatome vesicles + AUR; transient
Guillain-Barre	Sporadic, autoimmune, usually transient
Fowler's syndrome	Idiopathic retention in young females a/w polycystic ovary syndrome Raised resting EUS tone on EMG Failed relaxation of sphincter during voiding indicated by raised urethral pressure profile during voiding Hormones, topical nitrates and BoTox ineffective; SNS promising

Management

Life expectancy of a paraplegic normal; tetraplegic a/w 20% reduction. QOL normally distibuted as for general population

Principles:

Urodynamics mandatory
Preservation of renal function priority
Surgery avoided unless absolutely necessary
Most patients adequately managed with anticholinergics and CISC

(i) Detrusor overactivity

- Conservative
  - Bladder training
  - Timed voiding
  - Sheath/catheter
- Pharmacological
  - Anticholinergics
  - Intravesical capsaicin +/- LA
  - Intravesical resiniferatoxin
  - Intravesical botulinum toxin
- Surgical
  - Sacral nerve stimulation (dorsal root)
  - Posterior rhizotomy and anterior root stimulation (SARS)
  - Clam ileocystoplasty
  - Detrusor myomectomy
  - Ileal conduit urinary diversion

(ii) Detrusor underactivity

No role for parasympathomimetics (distigmine bromide a/w risk of cholinergic crisis)
Clean intermitent self catheterisation (CISC)

(iii) Sphincter overactivity
Clean intermittent self catheterisation
? Botulinum Toxin
Transurethral sphincterotomy

(iv) Sphincter underactivity
Pads, sheath or catheter
Ephedrine or pseudoephedrine (not duloxetine)
Bulking agents
Autologous sling/ TVT
Artificial sphincter
Bladder neck closure and mitrofanoff

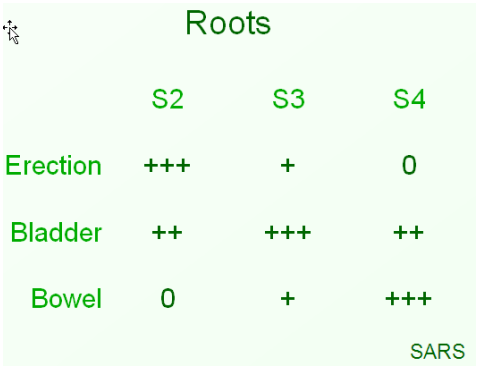
<u>Clean intermitent self catheterisation (CISC)</u>
Lapides 1972
Indicated for residual volumes > 100 ml
Good eyes and hands required
Careful counselling and assessment required – CNS

<u>Transurethral sphincterotomy</u>
Indications
Failed first-line therapy (anticholinergic/CISC) due to:
High voiding pressure /DSD
Hydronephrosis
VUR
Severe autonomic hyperreflexia
Typically performed under spinal to reduce risk of autonomic hyperreflexia
Long incision at 12 o'clock from bladder neck to bulbar urethra – can be difficult. Patient then managed by condom catheter

<u>Sacral nerve stimulation</u>
Anterior and posterior rami of sacral nerves *emerge separately* from the anterior and posterior sacral foraminae.
2 types:
Posterior stimulators
Medtronic ® InterStim Sacral Nerve Stimulation (SNS) System™, Minnesota, USA
Interfere with spinal reflex arcs by electrical modification at S3 posterior sacral foramina.
Used to suppress detrusor overactivity - identical to those for used for severe OAB
Useful for patients with incomplete suprasacral spinal cord lesions with some preserved sensation
Also appears to be effective for patients with Fowler's syndrome (non-obstructive urinary retention) – improves detrusor-sphincter co-ordination, possibly by reducing protective afferent signally from pelvic floor

Anterior root stimulation

Sacral anterior root stimulation (SARS)
Pioneered by Giles Brindley (Finetech-Brindley technique)
Electrodes applied intrathecally to anterior nerve root – much more extensive procedure involving laminectomy
Usually combined with posterior rhizotomy in patients with complete suprasacral spinal cord lesions
Posterior rhizotomy (sacral deafferation) abolishes bladder reflex, diminishes autonomic dysreflexia, improves capacity, reduces DSD, improves bowel continence, *but* loss of reflex erections, reflex ejaculation and bowel emptying
Different nerve roots for different somatic motor loss:



Very effective, but high morbidity, high reoperation rate and implant dysfunction